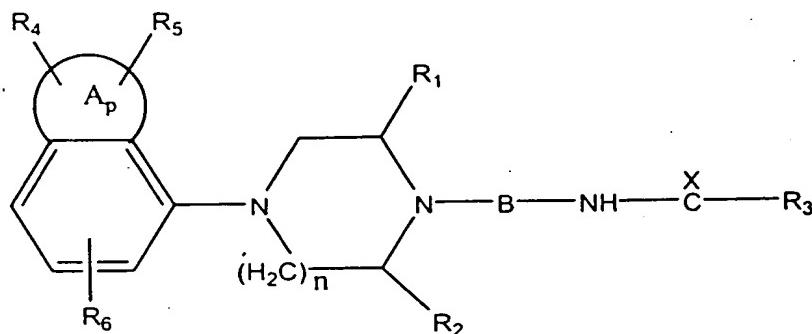


WE CLAIM:

1. A method of treating Attention-Deficit/Hyperactivity Disorder ("ADHD") in humans by administering a pharmaceutical formulation containing a therapeutically effective amount of a compound or compounds having full agonist or partial agonist activity at 5-HT<sub>1A</sub> receptors, wherein any non-5-HT<sub>1A</sub> agonist that is included in said formulation as an active ingredient, no such active ingredient is nicotine or a nicotinic agonist with the proviso that said formulation does not comprise nicotine or a nicotine agonist or buspirone or sunipetron.
2. A method according to claim 1 wherein 5-HT<sub>1A</sub> agonist is a compound according to the formula I:



I

wherein

- R<sub>1</sub> and R<sub>2</sub> independently of each other represent hydrogen or an alkyl having 1-3 carbon atoms;
- R<sub>3</sub> is an aryl group or heteroaryl group which may be substituted with one or more substituents selected from the group consisting of halogen, trifluoromethyl, nitrile, nitro, alkoxy, having 1-3 carbon atoms, hydroxy, esterified hydroxy, and alkyl having 1 or 2 carbon atoms;
- X is O, S, or NH;
- B is the group -CH<sub>2</sub>-CH<sub>2</sub>- or -CH(CH<sub>3</sub>)-CH<sub>2</sub>-;

- n has the value 0 or 1;

- p has the value 0 or 1;

-- where p has the value 1,

A is O-CH<sub>3</sub>, or forms, with the two carbon atoms of the phenyl group, an optionally substituted, entirely or partly unsaturated, cyclic group having 5-7 atoms in the ring, which comprises 1-3 hetero atoms from the group O, S, and N, with the proviso that the sum of the number of oxygen and sulfur atoms is at most two,  
--- and where A is not O-CH<sub>3</sub>,

R<sub>4</sub> is hydrogen or straight or branched chain alkyl having 1-3 carbon atoms and R<sub>5</sub> is hydrogen, halogen, alkyl having 1-3 carbon atoms, methylene, ethyldiene or vinyl, a straight or branched hydroxyalkyl group having 1-3 carbon atoms, which may be etherified or esterified, or an alkyl branched hydroxyalkyl group having 1-3 carbon atoms in the straight or branched alkyl group, an oxo group or a phenyl group; and

- R<sub>6</sub> is a hydrogen or fluoro atom;

wherein

- the compound may be a racemate or a single diastereomer or enantiomer;  
- or a pharmaceutically acceptable acid addition salt thereof.

### 3. The method of claim 2,

wherein

- R<sub>1</sub>, R<sub>2</sub>, and R<sub>6</sub> are hydrogen;

- R<sub>3</sub>, is a lipophilic aromatic alkyl, selected from the group consisting of benzene, halogenated benzene, cyclohexane, and 2-thiophene;

- X is O, S, or NH;

- B is the group -CH<sub>2</sub>-CH<sub>2</sub>-

- n has the value 1; and

- p has the value 0 or 1,

-- and where p has the value 1.

A is O-CH<sub>3</sub>, or forms, with the two carbon atoms of the phenyl group, an optionally substituted benzodioxane, a hydroxyalkyl having 1-2 carbon atoms, or a furan, R<sub>3</sub>,  
--- and where A is not O-CH<sub>3</sub>,  
R<sub>4</sub> is hydrogen, and  
R<sub>5</sub> is hydrogen, or chiral -CH<sub>2</sub>OH- at the 2 position of the benzodioxane ring.

4. The method according to claim 3, wherein the compound is flesinoxan, wherein

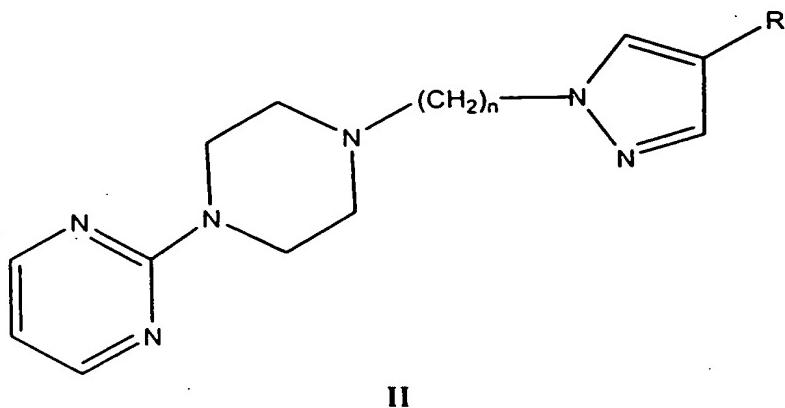
-R<sub>1</sub>, R<sub>2</sub>, and R<sub>6</sub> are hydrogen;  
-R<sub>3</sub> is halogenated benzene group, having a fluoro in the para position;  
-X is O;  
-B is the group -CH<sub>2</sub>-CH<sub>2</sub>-;  
-n has the value 1;  
-p has the value 1;  
-A is benzodioxane;  
-R<sub>4</sub> is hydrogen  
-R<sub>5</sub> is chiral -CH<sub>2</sub>OH- at the 2 position of the benzodioxane ring; and  
-the salt is hydrochloride.

5. The method according to claim 4, wherein the compound is administered at a dose of approximately 0.04 mg/day to 4 mg/day.

6. The method according to claim 5, wherein the compound is administered at a dose of approximately 0.1 mg/day and 1 mg/day.

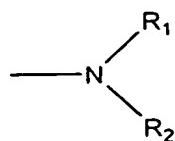
7. The method according to claim 6, wherein the compound is administered at a dose of approximately 0.1 mg/day and 0.5 mg/day.

8. The method according to claim 1, wherein the 5-HT<sub>1A</sub> agonist is a compound having the formula II:



wherein

- n can have the value 1 to 6;
- R is a hydrogen, a halogen, a lower alkyl radical having 1-4 carbon atoms, a heteroaryl radical, a sulpho radical, an N-substituted or N,N-disubstituted sulphamoyl radical, a nitro radical, a hydroxyl radical, an oxo radical, a lower alkoxyradical having 1-4 carbon atoms, a cyano radical, a lower alkylcarboxylate radical having 1-4 carbon atoms, an aryl or substituted aryl radical, or an amino or substituted amino radical of formula



in which R<sub>1</sub> and R<sub>2</sub>, independently are a hydrogen, an alkyl radical, an aryl radical, an alkylcarbonyl radical, an arylcarbonyl radical, an alkylsulphonyl radical or an arylsulphonyl radical, the alkyl fragments of these radicals containing from 1-4 carbon atoms; and

wherein

- the compound may be a racemate or a single diastereomer or enantiomer;

-or a pharmaceutically acceptable acid addition salt thereof.

9. The method according to claim 8, wherein the compound is lesopitron, wherein

- n is 4,
- R is chloro; and
- the salt is dihydrochloride.

10. The method according to claim 1, wherein the 5-HT<sub>1A</sub> agonist is selected from the group consisting of flesinoxan, lesopitron, BAY x 3702, F11440, LY228729, LY293284, NAE-086, S14506, S14671, S16924, or gepirone.

11. The method according to claim 1, wherein the 5-HT<sub>1A</sub> agonist(s) is the sole ADHD active component(s) of the formulation.

12. The method according to claim 10, wherein the 5-HT<sub>1A</sub> agonist is administered at a dose of approximately 0.01 mg/day to 100 mg/day.

13. The method according to claim 10, wherein the 5-HT<sub>1A</sub> agonist is administered at a dose of approximately 0.1 mg/day and 10 mg/day.

14. The method according to claim 10, wherein the 5-HT<sub>1A</sub> agonist is administered at a dose of approximately 0.1 mg/day and 2 mg/day.

15. The method according to claim 1, wherein the intrinsic activity of the 5-HT<sub>1A</sub> agonist is at least 0.5-1.0.

16. The method according to claim 15, wherein the intrinsic activity of the 5-HT<sub>1A</sub> agonist is at least about 0.6-1.0.

17. The method according to claim 16, wherein the intrinsic activity of the 5-HT<sub>1A</sub> agonist is at least about 0.7-1.0.

18. The method according to claim 17, wherein the intrinsic activity of the 5-HT<sub>1A</sub> agonist is at least about 0.8-1.0.

19. The method according to claim 2, wherein the difference in affinity of the 5-HT<sub>1A</sub> agonist for 5-HT<sub>1A</sub> receptors compared to any of 5-HT<sub>1B/1D</sub>, 5-HT<sub>2</sub>, D<sub>2</sub>, D<sub>4</sub>, α<sub>1</sub> or α<sub>2</sub> receptors or SERT, DAT, or NET ( $ΔpK_i$ ) is at least 1.
20. The method according to claim 2, wherein the difference in affinity of the 5-HT<sub>1A</sub> agonist for 5-HT<sub>1A</sub> compared to D<sub>2</sub> receptors ( $ΔpK_i$ ) is at least about 2.
21. The method according to claim 2, wherein the difference in affinity of the 5-HT<sub>1A</sub> agonist for 5-HT<sub>1A</sub> receptors compared to any of 5-HT<sub>1B/1D</sub>, 5-HT<sub>2</sub>, D<sub>2</sub>, D<sub>4</sub>, α<sub>1</sub> or α<sub>2</sub> receptors or SERT, DAT, or NET ( $ΔpK_i$ ) is at least about 2.